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CHILDREN'S HOSPITAL

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VOLUME V NUMBER 11

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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

13th and W Streets, Washington 9, D. C.

Vol. V *October 1949* No. 11

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STUDIES ON BED ROCKING, HEAD BANGING AND RELATED RHYTHMIC PATTERNS

SPECIAL REPORT

Reginald S. Lourie, M.D.

If we follow the maturational process in newborns as they develop cephalocaudad motor control, we see a certain number of them, usually healthy in every discernible way, supplying their own rhythmic motor patterns. By the time they are 2 to 3 months of age, a few of them are rocking their own heads, the only part of their bodies over which they have some voluntary control. By the time they are 6 to 10 months of age, more of them are rocking their heads and a few others have gone into more dramatic forms of rhythmic activity such as banging thier heads actively against their crib headboards or getting up on their hands and knees and rocking rhythmically back and forth.

This study reports the observations in 130 such children. The youngest child encountered in this type of activity began it at 1 month of age—the latest onset was 2 years. About 5% more boys are involved than girls. In all these children these movements were definitely voluntary and without nystagmus, appearing to be thus differentiated from spasmus nutans and similar involuntary head movements.

With some of the children these rhythmic movements are transitory. In others they remain for months, or even years. In an unselected pediatric clinic population 15% to 20% of the children had rocked, banged, or swayed in one form or another for a longer or shorter time. In private practice the figure is smaller, 10%. In the 5% of children in which these patterns last longer they go on usually until the child is 2½ to 3 years old. Occasionally, however, we see it prolonged much past this point. We have seen it persisting in pure form in children up to 12 years of age. In the older children this rocking or swaying behavior is in some cases continuous from infancy, while others return to it under varying conditions. But in the vast majority, as the children have gotten older, these larger rhythmic patterns have changed in form instead of being dropped completely.

Sooner or later the children appear to realize that the adults around them are concerned about these motor activities. The adults want to do something about the hair that's rubbed off by the head roller and the bruises and callouses of the head banger. The noise made by the crib as it is rocked across the floor has had considerable nuisance value such as broken leases and irritable neighbors. Many pressures are put on these children to modify these activities. Many ingenious contrivances have been devised to harness

the disturbing and supposedly aimless movements, sometimes justifiably, to allow the rest of the family a night's sleep. In spite of the ineffectiveness of such restraints and devices the prognosis is better than it is for snoring. The children either eventually decide to conform to the wishes of their elders, no longer need the larger rhythmic patterns, or else they substitute other patterns for them. We see a variety of partializations of the original rhythmic forms of isolation of the movement to one part of the body. Examples are rhythmic toe curling, tooth grinding, ear pulling, finger tapping, nose rubbing, and skin scratching, among many possible variations. Many of these substitutes can be carried into adult life, often acquiring other uses and values. Other repetitive activity forms which appear about the time of spontaneous disappearance of rocking and swaying are stereotyped play patterns.

CONSIDERATIONS ON CLINICAL VARIETIES OF CHILDHOOD RHYTHMIC PATTERNS

When we examine these motor phenomena of a repetitive nature in early childhood we see them as apparently having a common denominator at their onset and for a varying period afterward. In origin they appear to represent an attempt to experience movement for the kinesthetic sensations that play an important rôle in the infant's development[1]. These rhythmic activities seem at this point to provide a means for helping the infant in its mobilization to achieve control. There are apparently wide variations in the constitutional needs of different children for such movement play and experience, and it seems to be more marked in children whose control over motility is less easily developed. In some cases there seems to be a familial tendency to this type of movement.

Clinically, the most common time for the onset and use of rhythmic motor patterns is when an infant is in the transition between one stage of growth and development and the next. A classical instance is the child who can only sit but is struggling with a drive to stand which it cannot carry through. At this point it may be found either beginning, or having an exacerbation of, rhythmic body movements. These often become diminished or drop out completely after maturation has proceeded far enough to allow it to stand. When this same child is ready to take its first step out on its own but keeps falling, then rocking, shaking, or head rolling may recur or again become more active. This phenomenon may occur too in the face of deprivation. At any point in its course a secondary value may become prominent, and the repetitive movement then seems to take on the nature of an additional need or satisfaction in addition to its organizing and mobilizing effect.

In some infants these rhythmic movements seem to have a predominantly

pleasure value. The 6 month infant who sways, rocks, or shakes when music plays has been described as obtaining movement pleasure[2]. This is seen in older children too in the face of lack of play outlets, monotony of interest and life, as in orphanages and institutions for mental defectives. In addition to the "pleasure from movement" factor there are a number of children who only rock when they are pleased with what they have accomplished, such as when they have had a particularly satisfying meal, are praised, are successful, admired, or having pleasant thoughts.

Probably in the over-all picture of their use the most widespread function of the rhythmic motor patterns in children is to express and relieve tension and anxiety. Mahler[3] has observed that motor release forms an always available safety valve against anxiety. While the origins of the rocking and other rhythmic forms observed in this series usually are not in tension-producing situations, they are often used sooner or later to announce and/or try to relieve apprehension, dissatisfactions, anger, threats, boredom, pain and frustration. Where this secondary value has become prominent, it is the chief cause of their persistence past one year of age. Their correlations with thumbsucking in this regard are striking. Indeed, thumbsucking at times is interchangeable with bed rocking, etc. The greatest use of these manifestations in relieving tension is as a prelude to sleep. If awakened, the infant who uses this "relaxational expedient"[4] must rock its body or roll its head to fall asleep again.

This tension-relieving or expressing rôle seems to be closely related to another function served by these movements, that of supplying compensatory satisfaction. This is true in children whose movement is restricted, as Levy has shown in his studies on movement restraint. Many forms of stereotyped rocking or swaying movements are found in children confined by illness or in cribs, playpens, tied down in hospital beds, etc. Interesting correlations have been made between these patterns in children and the headshaking behavior of closely confined hens, the weaving tics of horses confined to their stalls, the upsweeping head movements of caged bears[5]. Illness in particular may initiate rhythmic movement forms. Children with chronic limiting illnesses such as severe congenital heart disease are not unusually found to resort to this type of activity in the absence of any other. Acute illness may foster a return to previously present but long gone rocking or swaying.

This principle applies equally as well to children whose motility is constricted or disturbed on an intrinsic basis as it does to those with environmentally imposed movement restrictions. Thus some infants with organic brain damage resulting in considerable loss of motility will adopt rhythmic patterns to the limit of their ability to move. Then too, the child who is able

to get around, but whose motility is awkward, distorted or frustrating, will seek compensatory motor satisfaction in stereotyped patterns of movement.

The same principle would seem to be applicable to children with "head-rolling rickets." The children presenting this symptom have, in addition to weakness, reportedly also shown bone changes which delay the acquisition of motor skills.

Similarly, children who are frustrated from achieving intellectual satisfactions have been seen to select this form of expression of dissatisfaction and/or compensatory satisfaction. Since mental defectives often have both inferior motility and ability to adjust it should not be surprising that many of them indulge in repetitive patterns of a motor nature, increasing in degree as they are increasingly unable to adapt successfully to the demands of their environments. Apparently in such defective children these manifestations can become very readily fixed and exaggerated as a form of satisfaction. In fact, it is so prominent in this group that some recent pediatric texts describe head rocking and banging as indicative of mental deficiency.

Characteristic of the entire group in which motility disturbances, confinement, frustration, and deprivation are prominent is the usual disappearance of the repetitive rocking or swaying and their substitutes, once the movement restraint is relieved, the motility is corrected, or better compensatory satisfactions are provided.

Another group in which these rhythmic body movements seem to serve a purpose includes older children with special problems limiting not only motility but also perception. A 7 year old boy with 70% to 80% loss of vision on a hereditary basis displayed a rhythmic swaying and whirling in preparation for carrying out requests or initiating any unfamiliar activity or game. Such whirling is reported to be not uncommon in blind children[6].

An 11 year old moderately severe cerebral spastic girl of normal intelligence, who had great speech difficulty, rhythmically rocked back and forth in situations where she particularly wanted to be clearly understood, and then she could come out with intelligible speech. One wonders whether in such cases the rhythmic movements do not supply a means of overflow of distracting and poorly directed motor impulses, providing a mobilizing factor much like the rhythmic patterns some stutterers use before they can get a word started.

One variety of rhythmic movement pattern which does not fit into any of the previous categories is the apparently normal infant who rocks only in its sleep. This form begins when the child is able to get up on its lands and knees or can sit up. It rarely persists beyond 2½ to 3 years of age. It is not a "relaxational expedient" in Gesell's terms, although some of these

children do rock themselves to sleep in addition. The sleep rocking begins sometimes hours after the child has fallen asleep and may continue for hours. It will stop if the child is awakened. (It does not depend on darkness as does spasmus nutans, because it occurs during daytime naps too.) More striking possibly than the rocker is the infant who bangs its head while sound asleep, in some cases sitting up. Some of these children seem to be not completely asleep.

Five cases, 2 girls and 3 boys, have come to attention in whom bed rocking and body swaying are the presenting or prominent symptoms later in childhood. In each of these children, ranging in age from 7 to 12 years, the persistent symptom had its roots in early childhood, but in 2 of them it had dropped out of sight for 2 and 4 years respectively.

DISCUSSION

There is no rhythmic experience which is limited to one form of sensation. Rhythmic experience is a grouping of auditory, kinesthetic, tactual, and visual stimuli. These are interrelated and depend on each other.

Titchener's extensive observations[7] led to his feeling that the existence of the kinesthetic sensation is primarily linked to auditory stimulation, probably chiefly due to contraction of the tensor tympani of the middle ear. This thesis seems to be borne out by the head banger in whom the auditory sensation produced by its activity is not only an integral part of the rhythmic movement but appears to be its most important component although the vestibular apparatus is also concerned. Following this premise a series of preliminary experiments were attempted. A metronome was set in action at the bedsides of 2 diurnal, purposeful head bangers and 2 children who banged their heads in their sleep. The children ranged in age from 12 months of 26 months. When the metronome was set at the same tempo that the child was using in its head banging it invariably stopped the activity. The sleeping children remained asleep but quiet. When the metronome was stopped, only the daytime head bangers sooner or later (3 to 15 minutes) resumed the head banging. When the metronome beat was slowed, one of the awake and consciously aware children returned to its own preferred rhythmic tempo. One of the sleeping children woke up. When the metronome was speeded up, they all stopped their movement but resumed it within 15 minutes after the superimposed rhythm was stopped. These maneuvers were also tried with one sleep rocker but he did not stop his rocking.

The extremely small numbers involved in these uncontrolled experiments would in themselves make them unreliable. It must also be considered that the supposedly substitute auditory stimulus could have had a predominantly distracting value. They are, however, mentioned here as a basis for

speculating on the rôle of the auditory experience in the head banger and to point a direction for further exploration.

Sherrington[8] felt that the ultimate basis of all rhythmic experience rests on a series of definite time units. Subjective time units or "mental time beats" have been postulated[9] and this is confirmed in the children studied here. In the great majority of the children who rock, roll, bang, or sway the pacemaker is the heart beat. Possibly this also has an auditory component since, as reported by Clausen, a great many individuals can distinctly hear their own pulses, with the sound localized to their ears. When the rhythmic movement is used for expression or relief of tension, anger, or any situation that will increase heart rate, the rate of rhythmic movement increases correspondingly but not always to the same extent as the rise in pulse. This is true not only for the body rocker or the head banger, but is also dramatically displayed by the older thumbsucker who, when disturbed, will suck its thumb furiously. The increased rate of the rhythmic activity seems not only to serve the purpose of stating and relieving the child's upset state, but also seems to serve a homeostatic function in that the rhythmic movement often has a retarding effect on the heart rate at such times.

In a minority of the children in this series the pacemaker is the breathing rate. The same acceleration with anger or stress and the secondary retarding action operates in these cases as is found where the pulse provides the subjective time unit. Possibly this phenomenon adds to the evidence of a physiological basis for Coleman's[10] clinical observation that rhythm of movement is the essential factor in development of endurance and postponement of fatigue.

At some time or other in its course most parents and many doctors equate the child's bed rocking and some of the other repetitive movements with genital masturbation. There is no clear evidence for this equation in the younger children in terms of the rhythmic activity being a substitute for genital masturbation. We can confirm Langford's observations that erections do not occur as part of such activity in boys.

It is only a relative minority of parents who become concerned about the varied rhythmic manifestations in their children, because if the rhythmic patterns do not get out of bounds or take on other values most parents seem to realize intuitively their normalcy. Where the repetitive movements have become more than annoying or reached symptom proportions, the problem of relieving them has been approached from 3 aspects. First, in those cases where it has been troublesome and appeared to have taken on values other than its normal ones in growth and development, an attempt has been made to replace the rhythmic movement with a rhythmic auditory stimulus (as described earlier) with the intent of interrupting the undesired activity. Results have been variable, but in a few cases it has been reported

by parents that sometimes, once the movement is replaced or substituted for by tapping or by using a metronome, the child does not resume the rocking that night. One parent bought a loud-ticking alarm clock, and reported that the bed rocking disappeared except for occasional recurrences.

A second and somewhat more fruitful approach has been to attempt to make the rhythmic movements purposeful. One mother put the swaying and rocking to music with reportedly good results in relieving the inconvenient rocking. Hobby horses, swings, see-saws, participation in rhythm bands, eurythmics, etc., have been prescribed for such children, but again with variable results. Of 10 children so approached parents reported definite decrease in frequency and intensity of the rhythmic activity in 2, and definite "cures" in 2 others. Since all 4 children were 18 to 28 months of age it must be considered that they were possibly ready to give up the rhythmic patterns in any case. However, in blind children this approach was almost specific in relieving the need to precede activity with swaying or whirling.

Probably the most constructive approach when these repetitive patterns are found is to use them as an indication that one should scrutinize the emotional climate and environment in which the child is functioning, looking particularly for constrictions and tension-producing conditions, as Langford suggests[11]. This is particularly true in children over 2½ years of age, including defective children.

Finally, after seeing how rhythmic motor patterns have been used by normal children in facilitating normal growth and development and also by handicapped children to help achieve better adaptation, the potentialities of the use of such rhythmic activities as therapeutic tools with disturbed children should be considered. The observations reported here on the blind and cerebral palsied children may indicate that similar approaches might be more extensively attempted with other handicapped children. Bender and Boas[12] have successfully used music and creative dance in the study, therapy, and training of deviate children. In some schizophrenic children, their rhythmic patterns offer one of the few means through which they can be approached by a therapist in establishing the relationships so necessary for treatment.

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ATYPICAL OSTEOMYELITIS

Case Report No. 161

Abram Berry, M.D.
Bjorn Gudbrandsson, M.D.

This is a report of seven cases of acute osteomyelitis in infants and children observed at the Children's Hospital during the year 1948. Five responded well to therapy, while the two who failed to respond developed bone changes while they were receiving what was thought to be adequate treatment.

Osteomyelitic bone changes were demonstrated by x-ray in all cases. Of three positive blood cultures obtained, hemolytic Staphylococcus aureus was isolated in two cases and a beta hemolytic streptococcus in the other. In one case, hemolytic Staphylococcus aureus was found in pus aspirated from a soft tissue abscess anterior to the involved bone. The youngest child was five weeks old. Four were under two years of age; the oldest was eight and one half years old. Four of them were negroes; three were white. The last one was a five month old male infant who was admitted with diarrhea and developed osteomyelitis after bone marrow infusion.

Case #1 T. B. 48-13632

T. B., a two and one half month old colored female was admitted to the hospital on December 4, 1948 because of a swelling of the left arm of two days' duration. She was perfectly well until two days prior to admission when a swelling was noted on the left arm, and at the same time she seemed to move the arm less.

The birth history and family history were negative.

Physical examination revealed a well nourished colored female infant with normal temperature. She did not move the left arm which she held in a 90° abduction with the elbow flexed in a right angle. Marked edematous swelling of the entire arm was observed and there were signs of a fracture at the upper end of the humerus. A swelling also was present on the upper right humerus and lower left tibia.

Laboratory findings were: hemoglobin, 12 grams; erythrocytes, 3,400,000; leukocytes, 13,700 with 34 per cent polymorphonuclears and 66 per cent lymphocytes. Serological tests for syphilis done on admission and five times during her hospitalization all were negative. The parents had a serological test done for syphilis and both were negative. Blood culture on admission and two days later grew out hemolytic Staphylococcus aureus, 12 colonies per cc., and the third one (after treatment had been

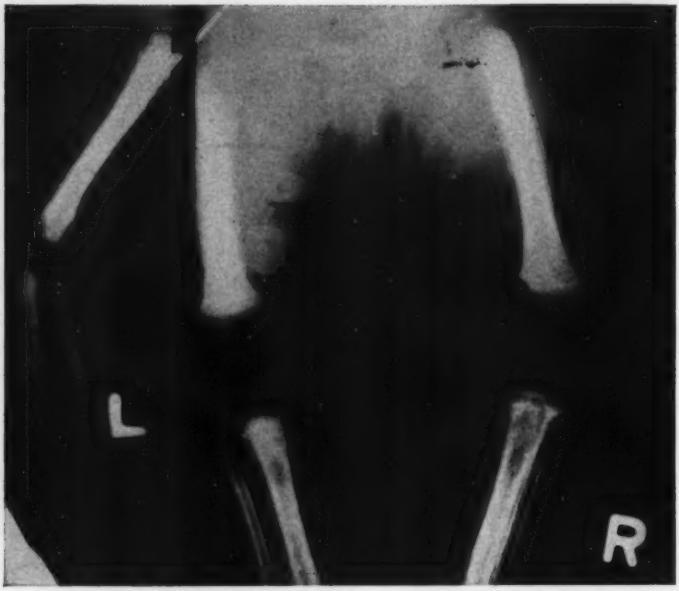

FIG. 1. T. B. Generalized osteomyelitis identical with changes found in luetic osteomyelitis.

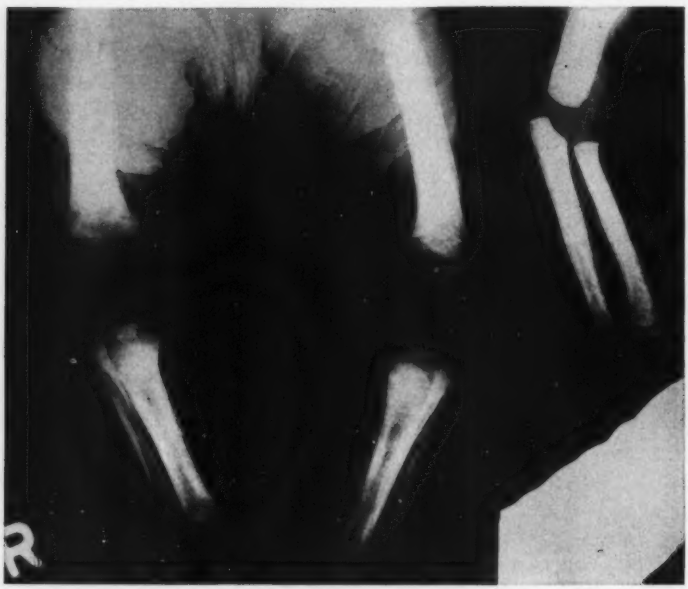

FIG. 2. T. B. One month later after therapy showing healing changes.

started) 7 colonies. The organism was sensitive to 0.1953 units of penicillin per cc. X-rays of the long bones showed generalized osteomyelitis with

FIG. 3. P. McC. Localized area of osteomyelitis jumping the epiphyseal space and involving the epiphysis.

FIG. 4. One month later showing healing of the epiphyseal involvement with improvement in the metaphyseal region.

considerable hemorrhage in both humeri. Luetic etiology was strongly suspected by the roentgenologist.

On the third hospital day, penicillin, 50,000 units, was administered every

three hours. This was continued for fourteen days. Sulfadiazine, one grain per pound per day, was given for thirteen days. The course was uneventful. Temperature remained normal and the oedema of the left arm gradually disappeared. She was discharged from the hospital in good condition. She has been following in the Out-Patient Department and clinically has been asymptomatic and x-rays showed marked improvement.

The great resemblance of the picture to congenital syphilis is of interest. The negative serological test and the positive blood cultures indicate that the bone changes undoubtedly were due to hemolytic Staphylococcus aureus.

Case #2 D. M. 48-6963

D. M., a sixteen months old white male, was admitted to the hospital on June 13, 1948 with the chief complaints of fever and drowsiness of five days' duration followed by tenderness in both legs. No treatment had been administered previous to admission. Physical examination on admission revealed a seriously ill child with temperature of 105°F. He was semicomatose. Respirations were not rapid. There were no localizing findings.

Laboratory results included: hemoglobin, 11 grams; erythrocytes, 3,600,000; leukocytes, 13,000 with 80 per cent neutrophils, 12 per cent lymphocytes, and 6 per cent monocytes. Urinalysis revealed 200 mgm. albumin and many erythrocytes and leukocytes; non-protein nitrogen was 64 mgm. per cent. Blood culture grew out beta hemolytic streptococci, 300 colonies per cc., and two days later grew out 44 colonies of the same organism. Subsequent cultures were all negative. The organisms were sensitive to 3.5 units of penicillin per cc.

The patient was given penicillin, 100,000 units every three hours, for seven days. Temperature came down to normal on his second hospital day. On the ninth day in the hospital, his fever rose again and penicillin was started again for two days. He was discharged on the sixteenth hospital day.

At home the parents noted that he did not use his left leg, and he was readmitted on July 1, 1948. During his first stay, x-rays of the pelvis and both femora were negative. X-ray of the same area at the time of the second admission showed a destructive process in the upper end of the left femur, and comparing these with previous films, it was evident that this process had developed during or after his previous treatment. He was now put on penicillin, 500,000 units every three hours for twenty-four days, and sulfadiazine, two grains per pound per day for fourteen days. A hip spica was applied.

The patient was discharged on the twenty-seventh hospital day in the cast. Clinically, he had been doing well with a normal temperature but the

process in the left femur had failed to show signs of healing. At the present time, after two more hospitalizations in August and September 1948, both for the purpose of changing the cast, he has now developed chronic osteomyelitis, and a surgical procedure is now planned to clean out the bone and remove the sequestrum.

Case #3 C. W. 48-6346

C. W., a five weeks old colored female, was admitted on June 1, 1948. The patient was well until four days prior to admission when the mother noted her child shaking her leg. Next day her right ankle and lower leg were swollen. She was seen in the dispensary two days prior to admission, and fever and tenderness were noted. X-ray of the leg was negative. Penicillin in oil, 150,000 units, was given. Physical examination revealed enlarged tender right inguinal nodes. The right leg was markedly swollen, and the skin was shiny, tense, red, and tender. The remainder of the physical examination was essentially negative.

Laboratory examination revealed 15,000 leukocytes with 40 per cent polymorphonuclears. Blood culture and Wassermann test were negative.

Penicillin was started on the second hospital day, 30,000 units every three hours, and hot compresses were applied locally. The subcutaneous tissue appeared fluctuant and incision and drainage was done on the third hospital day revealing a large quantity of bloody pus. Culture of the pus revealed hemolytic Staphylococcus aureus. X-ray on the sixth hospital day revealed an area of destruction of the distal end of the right tibia. Sulfadiazine, ½ tablet every six hours, was started on the eighth hospital day. X-rays on the fourteenth hospital day revealed increased destruction of the tibia. The leg was placed in a cast on the seventeenth hospital day. All therapy was discontinued on the twenty-fifth day and the patient was discharged. X-ray of the leg three weeks later revealed periosteal calcification with no evidence of activity. No further follow-up is available at this time.

Case #4 J. T. 44-7960

J. T., a five year old white male, was admitted on December 30, 1947. Ten days prior to admission, the patient jumped from a high place and began to complain of pain in the left foot. Subsequently, this pain became more severe and involved the ankle and calf of the leg. The ankle became swollen four days before admission and had not responded to local heat. X-rays taken on December 26 were negative.

Physical examination revealed the left ankle to be swollen, hot, and red. There was pain upon motion of the ankle. Left inguinal adenopathy was noted. The remainder of the physical examination was negative.

Laboratory examination revealed 6,800 leukocytes with 50 per cent

polymorphonuclears. Blood culture was negative. X-rays on admission revealed beginning destruction of the fibula on medial aspect.

The patient was placed on penicillin, 50,000 units every three hours, from the first to the twenty-fifth hospital day. Sulfadiazine, one grain per pound per day, was administered for six days.

X-rays on the fourteenth hospital day revealed some periosteal reaction and another taken on the twenty-first hospital day was reported as completely healed. Later films were reported as negative.

Case #5 B. A. B. 44-4359

B. A. B., a six year old white female, was admitted on December 21, 1947 with a complaint of pain in the right ankle and increasing difficulty in walking of three days' duration. Fever and redness of the ankle developed the day prior to admission. There was no history of injury.

Physical examination revealed an infected pharynx. The right ankle was swollen, red, and tender. Admission diagnosis was acute rheumatic fever.

Laboratory examination on admission revealed: leukocytes, 4,600 with 85 per cent polymorphonuclears. On the third hospital day, she had 13,600 leukocytes with 80 per cent polymorphonuclears. Sedimentation rate was 50 mm. per hour. A blood culture was negative.

The patient was treated with aspirin and codeine for two days without improvement. On the third hospital day, a tentative diagnosis of osteomyelitis was made and the patient was started on penicillin, 100,000 units every three hours, which was kept up for twenty-four days. X-ray taken on the sixth hospital day revealed an early destructive process of the distal head of the fibula. Repeated x-rays revealed calcification of the destruction with filling in of the defect. She was clinically recovered when discharged on the twenty-sixth hospital day. Follow-up film revealed complete healing of the destructive process.

Case #6 A. T. 43-3785-A

A. T., an eight and one half year old colored male, was admitted on June 8, 1948 with vague history of injury five days prior to admission. The next day he complained of pain in the left leg and it was noted that the left leg and foot were swollen. He grew steadily worse and became irrational the day prior to admission.

Physical examination revealed a dehydrated, irrational colored male critically ill. The left leg was swollen, hot, and tender. Slight pitting edema was noted. Inguinal glands on the left were enlarged and tender.

Laboratory examination on admission revealed 7,000 leukocytes with 82 per cent polymorphonuclears and on the fifth hospital day there were 18,000 leukocytes with 80 per cent polymorphonuclears. Blood culture

revealed H. Staphylococcus aureus, 246 colonies per cc. of blood, sensitive to 0.625 unit of penicillin. The remaining blood cultures were negative.

Continuous intravenous fluids were given for the first forty-eight hours in the hospital. Penicillin, 100,000 units every three hours, was instituted and on the third hospital day was increased to 200,000 units. This was done for sixteen days at which time duracillin, 300,000 units twice a day was administered. He continued on this therapy for fifty-four days. Sulfadiazine was started on the second hospital day.

On his sixth hospital day, fluctuation was noted over the medial surface of the left leg. An incision and drainage was performed and a considerable amount of pus was discharged. X-ray of the leg, which previously had been negative, now revealed early destruction of the tibia which has gradually increased in spite of prolonged and vigorous treatment. He was discharged on the ninty-ninth hospital day and follow-up x-ray revealed extensive destruction and sequestration of the tibia. He was re-admitted on January 21, 1949 for sequestrectomy.

Case #7 L. P. 48-12025

L. P., a five month old colored male, was admitted on December 13, 1948 because of diarrhea and bronchopneumonia. He was treated with bone marrow infusion and penicillin, 30,000 units every three hours. Bone marrow infusion was terminated on the second hospital day, when it had stayed in for twenty-four hours. On the third hospital day, swelling and pain were observed over the entire left leg. On his fourth day, penicillin was increased to 50,000 units every three hours, which was kept up until his thirtieth hospital day when it was changed to duracillin, 600,000 units daily, until his discharge on the thirty-ninth hospital day. X-rays taken on the fifteenth day in the hospital showed marked destruction of the upper third of the left tibia; another taken on discharge from the hospital showed some healing of the process with increased calcification. Clinically he was in good condition when discharged.

DISCUSSION

Acute hematogenous osteomyelitis was originally a surgical disease. During the past decade there has been a trend toward conservatism with the introduction of sulfa drugs and antibiotics. During that period, osteomyelitis in infants and children has changed from a severe disease with high mortality and high incidence of chronicity to one of almost no mortality and low chronicity.

Recent reports on the treatment of osteomyelitis reveal uniformly good results with penicillin in doses up to 50,000 units every three hours[1, 2]. In spite of this rosy picture painted by most authors some cases do not

respond to what is generally supposed to be thoroughly adequate therapy and go on to surgery. In this category, cases #2 and #6 are noted. They were admitted in the earliest stages of the disease, the former receiving 100,000 units of penicillin every three hours for ten days, and the latter receiving 200,000 units every three hours for sixteen days without stopping the progress of the bony lesions. In both of these cases, surgery had to be performed.

One of our cases (#6) was confused with acute rheumatic fever. This mistake may easily be made in the early stage of the disease, but the differentiation must be made promptly. The diagnosis was suspected early and proper treatment instituted. A prolonged trial of aspirin may be diagnostic in acute rheumatic fever, but would prove to be disastrous in acute osteomyelitis. Since penicillin would not interfere with the treatment of acute rheumatic fever, we feel that it would be advisable to use penicillin and sulfa in all cases in which the differentiation between the two is doubtful.

Case #7 developed osteomyelitis after a bone marrow infusion of twenty-four hours duration while receiving penicillin, 30,000 units every three hours. Heinild[3] et al report 1,000 cases of bone marrow infusion with only five cases of osteomyelitis, an incidence of 0.5 per cent. Rooney[4] reports 100 cases of bone marrow infusion with 2 per cent osteomyelitis. In spite of their good results, we feel that bone marrow infusions are not harmless procedures and should be used only in an emergency. In this regard, massive doses of penicillin should seem to be indicated to prevent the occurrence of this complication.

Dillhunt[5], Cass[6], and Hutter[7] report several cases of generalized osteomyelitis in newborn infants, which presented a varied picture from that seen in older ones. The disease is systemic, involving most of the long bones and is suspected by swelling of the extremities. The diagnosis is confirmed by positive blood cultures and positive x-ray findings in the involved bones. They point out that generalized osteomyelitis in infants often runs a mild course, systemic in nature, with a low grade of toxicity; it responds well to treatment and leaves practically no residue. Case #1 presented all of these findings. However, the bone changes by x-ray were confused with lues. With a negative family history for lues, repeated negative blood serology, positive blood cultures on three occasions, and typical response to treatment, the diagnosis of generalized osteomyelitis was made.

Three of our cases had positive blood cultures on admission and osteomyelitis was suspected on two of these. In case #2, adequate therapy for septicemia was given and the patient was discharged without any evidence of bone involvement, only to develop this at home. We feel that any case

of septicemia in infants and children should be considered as possible or potential osteomyelitis and that treatment and follow-up should be carried out with than in mind.

SUMMARY

A review of seven cases of osteomyelitis is presented, three of which had positive blood cultures. One case followed bone marrow infusion and one presented generalized bone findings. One case was confused on admission with acute rheumatic fever. All cases were treated vigorously with penicillin and sulfa; five cases showed marked improvement; two cases needed surgery.

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ACUTE POLYNEURITIS

Case Report No. 162

Elmer O. Bean, M.D. J. W. 49-3902

J. W., a six year old white female, was admitted to the hospital on March 24, 1949 with the chief complaint of inability to walk or stand of one day's duration. She had complained of pain in the toes of both feet on the day before, but this was not severe and had lasted only two hours. The morning of admission, the patient complained of transitory pain in the left knee and both ankles and she could not stand or walk unassisted. The evening of admission she stated that she had a "funny feeling" in her hands when she tried to hold a cracker. Later she could not raise a cup to her mouth, although she could raise it part way. Her parents noted that her finger grip was very weak.

There was no history of any infection in the past six months. Two days prior to admission, she had fallen out of a swing and landed on her back; however, she said that the fall was light and that she didn't even cry. There were no apparent sequelae following this. The child is one of five children (a twin) who is well-mannered and not spoiled. All of the siblings are treated equally. She is especially attached to her twin brother. The past history is essentially negative except for some asthma. She is allergic to horse serum. The family history is also essentially negative. A relative, Dr. Martin, supplied some additional information. Recently Jean and her playmates were known to have been playing with and dismantling an old battery. Also, a distant cousin had acute porphyria while in the service.

On admission the patient's temperature was 99.2°F., pulse 130, respirations 25 and blood pressure 128/60. She was very cooperative. The child could not elevate herself to a sitting position from the supine position. When sitting, she assumed a tripod position and complained of back pain on attempt to flex the trunk or the thighs. Examination of the eyes, ears, nose and throat were essentially negative. The fundi were normal. There was slight rigidity of the neck. The heart, lungs, and abdomen were negative. The skin was clear with no rashes or petechiae. The patient was examined for ticks. There was deltoid weakness on the left side, questionable bilateral biceps weakness, bilateral adductor weakness of the thighs and peroneal weakness, particularly on the left. The biceps and the abdominal reflexes were the only reflexes elicited on admission. The cranial nerves were intact and there was no clonus.

A spinal tap was done which revealed clear fluid, with an initial pressure of 160 mm. of water, a rise of 240 mm. of water on jugular compression,

with a subsequent fall to 120 mm. There were 8 white blood cells and 2 red blood cells, 10 mg. % of protein, 720 mg. % sugar and 710 mg. % chlorides. The urinalysis was essentially negative. The white blood count was 10,500 with 78% neutrophiles. No stippling of the red blood cells was noted.

On the second hospital day, the physical examination revealed the following findings: Loss of muscle tonus on the left side; weakness of the left deltoid and extensors of the left thigh; almost complete paralysis of both anterior tibial muscles; absence of all deep tendon reflexes; a questionable lateral nystagmus, dilated pupils which reacted to light and accommodation, and a normal fundoscopic examination; minimal intention tremors of the upper extremities with no gross dysmetria; inability to assume a sitting position from a supine position; inability to stand with the feet close together and inability to walk without support. A repeat spinal tap three days later was essentially as on admission except that there were four cells instead of eight.

On the fourth hospital day, the paralysis of the anterior tibial muscles assumed a spastic nature and there was pain on motion. There was a three-plus nuchal rigidity. The cranial nerves remained intact, and no sensory changes were noted.

X-rays of the skull, long bones, spines, and chest were essentially negative. An electrocardiogram on the 13th hospital day was within normal limits. Urine examination for porphyrin was negative and for lead within normal limits. Another spinal tap on the 9th hospital day revealed clear fluid, 10 mg. % protein, 45 mg. % sugar, with 12 cells (74% neutrophils and 28% lymphocytes). Spinal fluid culture and throat cultures for diphtheria were negative.

On the 13th hospital day, the patient began to slowly improve, the motion of the arms being regained rather quickly with slight improvement in the legs. The nuchal rigidity, however, was still marked and there were marked bilateral Kernig's. Physiotherapy was instituted, and on the 23rd hospital day she was able to walk unassisted about the ward. The hand muscles were gradually returning to normal when she was discharged on the 29th hospital day to be followed in the Physiotherapy Department.

Throughout her hospital stay, the child was pleasant and cooperative. Her course was essentially afebrile except for one rise to 101°F. Therapy was purely symptomatic.

DISCUSSION

John A. Washington, M.D.: The outstanding feature was a rapidly progressing, fairly generalized paralysis with loss of tendon reflection which spared the cranial nerves. The muscles of respiration also were spared so

that the child maintained a cheerful demeanor throughout her illness. Only when her neck or thighs were flexed passively did she complain of discomfort.

The following diseases were considered:

Poliomyelitis

Among the diseases considered this was the only one in which stiffness of the back and neck is characteristic. However, it seemed unlikely that poliomyelitis would cause this degree of sudden paralysis without producing fever, malaise, and spinal fluid findings.

Lead poisoning with neuritis rather than encephalitis

It seemed possible that the story of the child's having handled a battery casing was important. The absence of lead lines on x-ray pictures and stippling of red cells on smear together with the fact that the condition is extremely rare made this diagnosis less likely than some others.

Acute porphyria with peripheral neuritis

This was excluded by the failure to demonstrate porphyrinuria.

Acute febrile polyneuritis of unknown etiology (Ford).

While the presence of fever preceding or accompanying the onset is characteristic of this condition, it cannot be excluded as a diagnosis on this ground alone. This entity is not uncommon in pediatric hospitals, and I have seen it a half dozen times. It carries a good prognosis so long as the muscles of respiration are not involved. Sometimes the facial nerves are involved but cranial nerves are not otherwise affected. Ford expresses doubt as to whether or not the condition is etiologically distinct from Landry's ascending paralysis.

Landry's ascending paralysis

Clinically, this differs from acute febrile polyneuritis of unknown etiology in two respects. It is not ushered in by fever and the paralysis ascends so as to involve various muscle groups in an order corresponding to their representation in the spinal cord. In our case, the paralysis is described as having affected the feet in the morning and the hands by the evening of the same day. The prognosis in Landry's ascending paralysis is decided also by the degree of involvement of the respiratory muscles. In neither condition is there a disturbance of sensation. From pathologic studies, Landry's paralysis is classified as a myelitis rather than a neuritis. The spinal fluid is always normal.

SUMMARY

I believe that our case belongs in one of these last mentioned categories. As it seems likely that they are not etiologically different and that the latter is really a subgroup of the former, one might expect to find cases such as ours which fail to fall distinctly into one group or the other. Acute polyneuritis of unknown etiology would seem the best classification for purposes of reference. It is the diagnosis commonly used. This title seems to ignore the possibility that the pathologic process may well be a myelitis as demonstrated in autopsies of individuals dying of Landry's ascending paralysis. The fact that our case had stiffness of the back and neck suggests that some myelitis was present.

DILANTIN INTOXICATION

Case Report No. 163

John P. McGovern, M.D.
Robert E. Martin, M.D.

E. C. 38-1962

E. C., a twelve and one-half year old white female was admitted to Children's Hospital July 18, 1949 with the chief complaint of staggering and dizziness. Her present illness began at eighteen months of age with the first of three admissions to this hospital. At that time she had a generalized convulsive seizure that lasted three hours; a fever of 104°F.; and a hematuria and albuminuria which persisted through numerous consecutive urinalyses. She responded well to the administrations of parenteral fluids and after three weeks of hospitalization was discharged apparently well. She was not seen again at Children's Hospital until the age of five years when it was revealed that she had been having mild convulsive seizures since shortly after her first discharge from this hospital. During this period, she had as many as twenty petit-mal seizures in one day. However, there would be intervals for as long as two weeks during which time she would be free of attacks. Due to the fact that those seizures were becoming more frequent and that now some of the seizures were apparently grand-mal in type, she was brought into the out-patient department where treatment was begun with dilantin 0.1 gram daily. Although she was instructed to return to the out-patient department for periodic evaluation of her epileptic status, she was not seen again until she was admitted to this hospital two years later at the age of seven years. At that time she was having severe grand-mal seizures lasting as long as thirty minutes and followed by periods of somnolence of four to five hour's duration. She was having approximately three to four such grand-mal seizures and eight to ten petit-mal seizures each month. While in the hospital, she was regulated with dilantin 0.1 gram two times daily and remained on this dosage following discharge from the hospital. Six months before the present admission, because of the increasing frequency and severity of the seizures, the dilantin dosage was increased to 0.1 gram three times each day with the addition of phenobarbital 0.05 gram three times each day. From that time until the present hospital admission, she was maintained on this dosage and had few seizures.

Ten days before the present hospital admission, she was seen in the clinic with a history of having had no seizures during the previous twenty days but with the complaint of constant drowziness. In order to alleviate the drowziness, the phenobarbital was reduced to two 0.05 gram tablets daily,

this reduction being compensated for by an increase in dilantin dosage to five 0.1 gram capsules each day. Three days later or seven days before the present admission, the child began complaining of headache, dizziness, and diplopia. The mother noticed a staggering gait and abnormal movements of the child's eyes. All of these symptoms increased in severity up to the time of admission.

The family history and past history were not remarkable. There was no family history of epilepsy, nor were any developmental aberrations noted in the patient.

The pertinent physical findings on admission revealed the patient to be alert, cooperative, in contact with her surroundings, but complaining of headache and dizziness. Examination of the skin revealed a sparse growth of coarse black hair in the regions of the upper lip, chin, and in the intercanthal area of the nose and brow.

The pupils were normal. Marked nystagmus was present in all quadrants with the rapid component in the direction of the gaze. Diplopia was present for objects held in the periphery of the visual fields. Fundiscopic examination failed to reveal any abnormalities. There was marked hypertrophy of the gums, the margins extending over one-half the normal tooth surface. They were rather spongy and bled easily with slight trauma. The neurological examination encompassed the remainder of the noteworthy physical findings. There was a fine tremor of the extended fingers. The reflexes were physiological throughout with the exception of absent abdominal reflexes in the left side. There was a positive Rhomberg with swaying then falling to the left. The gait was ataxic with a tendency to deviate to the left. The cerebellar tests of finger to nose and heel down shin, however, were well performed.

Lumbar puncture revealed normal manometric readings and no increase in cells or protein in the spinal fluid. The hemogram was within normal limits as was the routine urinalysis. A test for barbiturate derivatives in the urine (modification of Koppanyi's Method)[1] was strongly positive.

The patient's course in the hospital was characterized by the rapid disappearance of symptoms following discontinuance of all medication. Within forty-eight hours after admission, headache and dizziness had disappeared. The gait was less ataxic, the Rhomberg test positive but less pronounced, and there remained only a very fine nystagmus on lateral gaze. The Koppanyi test at this time was weakly positive. Ninety-six hours after admission all signs and symptoms had disappeared and the urine was now negative for barbiturate derivatives. The patient was observed for four more days with no recurrence of symptoms. She had no convulsive seizures during her stay in the hospital.

DISCUSSION

Sodium diphenyl hydantionate (Dilantin), a drug closely related to Nirvanol, was first extensively studied as an anticonvulsant by Merritt and Putnam[2] in 1938. Since that time it has proven to be most useful in the treatment of epilepsy, but with one major disadvantage—it is toxic for some individuals even in relatively small doses. Toxic reactions from dilantin are of many types and although mild, occur in approximately 15 per cent[3] of patients receiving the drug. As the complications are of many types, and as an occasional patient may show symptoms in more than one physiologic segment, in the interest of clarity, these complications will be discussed under the various organs involved.

Oral Complications. The most frequent toxic reaction to dilantin therapy is the development of hyperplasia of the gums. This is most commonly seen in children or in young adults, being noted by Kimball[4] in 68 out of 119 epileptic children receiving dilantin. The gums return to normal with no treatment other than cessation of the drug although Robinson[5] reported one case in which the hypertrophied tissue remained after the drug was discontinued.

Cutaneous Complications. Complications involving the skin are among the most common toxic signs, being present in about 5 per cent of patients at some time while receiving this medication. The cutaneous eruptions are usually of a mild generalized erythematous type, but may show hemorrhagic manifestations. Most of these rashes disappear regardless of whether or not dilantin is continued. A case of dermatitis exfoliativa[6] and a fatal case of hemorrhagic erythema multiforme[7] have been reported. Hypertrichosis about the face and extremities has been observed in adolescent girls.

Gastro-intestinal Complications. Dilantin is a strongly alkaline preparation. The gastro-intestinal symptoms of nausea, epigastric distress, and in some instances severe vomiting, are thought to be due to the ingestion of this basic substance on an empty stomach. This complication is avoided if the drug is taken with food either just before, during, or just after eating.

Central Nervous System Complications. Toxic reactions to dilantin, with manifestations referable to the central nervous system, tend to appear between the third and tenth day of treatment. The symptoms are usually mild and transitory in type; most often consisting of heacaches, diplopia, tremors, vertigo, and ataxia. Reduction in dosage of the drug usually brings prompt relief. Harris and Ewalt[8] report a case with generalized symptoms and localizing signs so typical of a brain tumor that a craniotomy was performed to explore the right frontal lobe. Hemiplegia[9] and toxic amblyopia[10] have also been reported as complications, the latter occurring in a twenty-seven year old male after taking dilantin in a dose of 0.1 gram

three times a day for four months. Visual acuity returned after withdrawal of the drug.

Complications of the Hemopoietic System. A slight leukocytosis and a mild eosinophilia are often encountered, but are of no major importance. Agranulocytosis[11] has been reported after the ingestion of 12.7 grams of dilantin over a period of sixty-seven days.

There are, in addition to the complications already mentioned, several toxic manifestations of lesser note. Finkelman and Arieff[12] report evidence of myocardial damage and demonstrate electrocardiographic changes, consisting chiefly of a prolongation of the P-R interval and/or a decrease in the amplitude of the T-wave. Increased libido[13] has been observed as a complicating factor. Epistaxis[14] has also been mentioned as a complication of dilantin therapy.

SUMMARY

1. A case of dilantin intoxication with an unusual combination of central nervous system manifestations associated with hirsutism and gum hypertrophy is presented.

2. A discussion with notation of pertinent articles relating to dilantin intoxication is included.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: William M. Crowell, M.D.
Francis J. Troendle, M.D.
By invitation: Allen B. Coleman, M.D.

William M. Crowell, M.D. R. L. S. 43-9553

The patient was an eight year old colored female who was admitted to Children's Hospital on December 9 and died the following May 27.

She was last well in June or July at which time she developed joint pains with swelling, regional edema, and low grade fever, all of which were intermittent. On October 23 she was hospitalized at another hospital with the above complaints plus the story of having had generalized chest and precordial pain for past three weeks with axillary and inguinal lymphadenopathy for the same time. On admission the following points were noted:

(a) well developed, well nourished, alert, and coöperative

(b) cheek discoloration of butterfly distribution

(c) generalized lymphadenopathy of discrete, non-tender nodes

(d) breath sounds suppressed and resonance impaired at left base posteriorly and in left axilla below sixth rib

(e) apical heart sounds distant but without thrills or murmurs

(f) abdomen full with eversion of umbilicus, shifting dullness in flanks and liver edge palpable one finger—breath below costal margin

(g) one plus edema of hands and feet

(h) pain in left elbow joint on passive motion

(i) temperature 101°F

By November 21, the patient was asymptomatic and was discharged, having made a spontaneous recovery. At this time her superficial lymph nodes were smaller but still palpably enlarged. Her positive hospital findings were:

(a) widening of upper mediastinum with a moderate increase of transverse cardiac diameter (a pericardial tap was negative) which persisted

(b) positive PPD #2

(c) a hemogram showed 3.86 million red blood cells, 9 grams of hemoglobin, 14,050 white blood cells with 87 per cent neutrophiles

(d) a heterophile antibody test was positive 1:448 (District of Columbia Health Department)

(e) negative or normal results were obtained for the following: urinalysis, Hinton and Eagle, erythrocyte sedimentation rate, non-protein nitrogen, total serum protein, venous pressure, circulation time, blood

cultures, trichinosis complement fixation and skin test, twenty-four hour sickle cell preparation, an electrocardiogram, and febrile agglutinations.

The patient was well for two weeks after her first hospitalization and developed a rash on her hands and posterior trunk. A few days after this, she developed generalized edema and hemoptysis.

On admission to this hospital of December 9, the presenting complaint was blood in the urine. The child was found to be mildly febrile, well nourished, and in no acute distress; however, the face, abdomen, and lower extremities were edematous, there were râles in the left base posteriorly, and the heart was enlarged to percussion to the left. O.T. 0.1 mg. and P.P.D. #1 were positive on admission. From December 14 to 24 and from January 13 to 20, the temperature spiked daily to 102–103°F. Urine cultures at these times showed diptheriods. The patient did not appear as ill as the temperature indicated. Sulfadiazine and sulfathiozole were used. From the time of these episodes until May 3 there were few intermittent temperature elevations, but for the most part the temperature was normal. A septic course began on May 3 for no apparent reason, and ended with the patient's death on May 27. The patient weighed 54 pounds on admission and approximately the same at death.

During the entire admission, the child appeared well but the physical examination showed intermittent migratory joint pains, an enlarged heart, facial, abdominal and pretibial edema, a generalized non-tender lymphadenopathy, intermittent basal rales in the chest, and transient rashes described as being erythematous reticulum-like macules or papules which could not be stimulated or reproduced by ultra violet irradiation. The abdominal distension became very painful before death.

A cardiac consultation on December 30 indicated that the patient was not in congestive heart failure as was thought on admission. A dermatological consultation on March 17 suggested that the rash resembled a contact dermatitis.

Two abdominal paracenteses were performed; the first on May 25, with 750 cc. of normal fluid being obtained, the second on May 26, with 1200 cc. of fluid being obtained. Cultures showed E. coli and Streptococcus hemolyticus respectively.

Three electrocardiograms were done, the first of which showed an "abnormal myocardium" and the others were apparently normal.

Chest x-rays showed an enlarged heart and increase in pulmonary fibrosis on the left side especially. An infiltrating lesion was noted at the first anterior right interspace, probably inflammatory. An intravenous urogram and bone x-rays were negative.

The heterophile antibody agglutination was questionably positive in 1:56 dilution on April 21, having been positive in a 1:28 dilution on January 25.

Febrile agglutinations were negative. All Wassermanns done were very high in titer but the Kahns done, even though positive, were of low titer. A cerebrospinal fluid serology was negative. A colloidal gold was 2221110000. Bacteriological examinations showed Staphylococcus albus, diphtheroids, Streptococcus anhemolyticus in a throat culture, a negative blood culture, a negative lymph node culture, and a negative cisternal fluid culture. Vaginal smears and cultures were negative. A lymph node biopsy showed chronic hyperplasia.

On April 20 and May 9, two non-protein nitrogen determinations were 53 mg. per cent and 57 mg. per cent, respectively. On January 25, total serum proteins were 6.69 per cent. On March 9, blood chlorides were 429 mg.; total proteins, 7.40 per cent and cholesterol, 143 mg.

Corrected erythrocyte sedimentation rates were 27 mm., 11 mm., 43 mm., and 42 mm. respectively for December 15 and 16, January 20, and March 3.

Blood counts were as follows: red blood cells 2.70 to 3.70 million averaging 3.2 million; hemoglobin 8 to 11 grams averaging 10 grams; white blood cells 7,000 to 14,000 with high of 36,700 and 44,400 on May 9 and May 22 respectively returning to 14,200 on May 25, the date of the last blood count. The differential white count averaged 60 to 80 per-cent neutrophiles of which 65 to 70 per cent were segmented.

Routine urinalyses were on an average as follows: specific gravity 1.002 to 1.027, albumen; 0 to 2000 mg. averaging 500 mg., sugar; negative acetone, negative, full range of pH; and microscopic occasional to many red and white blood cells. A Mosenthal concentration test was within normal limits.

DISCUSSION

Allan B. Coleman, M.D.: This long involved case can be summarized as that of an eight year old negro female with a year-long illness, terminating fatally, and involving, at various times, the joints, the heart, the kidneys, the serous body cavities, the lymph nodes, and the skin, all with intermittent or remittent fever.

In an attempt to make a single diagnosis fit all of this widespread involvement, I shall consider the patient as a whole, rather than system by system.

The laboratory data seems less than helpful; I am unable to reconcile the heterophile tests results with any diagnosis which I would be willing to defend, and have been forced to discard it in my thinking. I know of no form of acquired syphilis in a child (and it would have to be acquired if we can believe the initial negative serology) which will produce such widespread visceral disease in so short a time.

I have been forced to eliminate as diagnoses such chronic infections as brucellosis and subacute bacterial endocarditis for lack of confirmatory

clinical and laboratory evidence, since we are presumably expected to make the diagnosis from the material presented in the protocol.

Could this child have lived with, and finally died of, severe rheumatic fever? Possibly so, but the cardiac consultant tells us that there was no congestive heart failure; and where is the murmur if this heart is severely diseased by rheumatic infection? Also, the renal pathology evidenced by hematuria, persistent massive albuminuria, and laboratory evidence of renal failure, and the syndrome of polyserositis are not easily explainable on this basis.

The x-ray films we have here are less impressive than the description of them in the case presentation; the only lesion with an inflammatory appearance present in the lungs was quite transient. Still, the diagnosis of some unusual and generalized form of tuberculosis must be given strong consideration. It could explain the fever, the pericarditis, the ascites, and the renal involvement; however, the perfectly normal excretory urogram that we see here spoils the case for tuberculosis, to some extent.

By process of elimination, we are forced now to consider that group of diseases characterized by widespread visceral disease and involvement of the skin and lymphatic structures, and which are to the pathologist generalized diseases of the vascular system. Among these, are dermatomyositis, periarteritis nodosa, and disseminated lupus erythematosis with visceral involvement known as Libman-Sacks syndrome. Although dermatomyositis cannot be diagnosed here because of the lack of involvement of the muscles, the entire picture fits the course of either of the latter two diseases quite well. A disease in this group will be my primary diagnosis; although periarteritis is said to be exceedingly rare in negroes, I shall put it first, since we have no evidence in the protocol of the rheumatic-like valvular heart disease which is usually present with the Libman-Sacks syndrome.

1. Periarteritis nodosa
2. Libman-Sacks syndrome
3. Tuberculosis, generalized

PATHOLOGIC DISCUSSION

E. Clarence Rice, M.D.: During life this patient attracted more than usual interest from the physicians who saw her and she was the subject of discussion in one of our Sunday morning conferences. The clinical impression was that that we were dealing with either disseminated lupus erythematosis, syphilis and/or chronic glomerular nephritis. The first diagnosis was most commonly held.

At necropsy the body was that of a well developed and nourished colored female with enlarged axillary lymph nodes and distended abdomen.

The pleural cavities were free of fluid and adhesions. The pericardial

sac contained a normal amount of clear yellow fluid and the pericardial surfaces were lightly adherent over the right ventricle. The epicardium was injected. The peritoneal cavity contained 400 ml. of cloudy foul smelling fluid from which hemolytic streptococci were cultured. The peritoneal surfaces were covered by a yellowish purulent exudate and the peritoneum was hyperemic. Loops of bowel were lightly adherent to each other.

The lungs were heavier than normal and mottled in appearance, the cut surfaces being similar in appearance with dark red areas which were rather firm. Microscopically the pleura was thickened and the arterioles showed some proliferation of connective tissue within their walls.

The bronchial and mediastinal lymph nodes were enlarged and hyperemic. Microscopically prominent reticulum was noted along with connective tissue proliferation and hyalinization of the blood vessels.

The heart weight 160 grams (normal—110 gm.) and measured 11.5 x 6.5 cm. and extended 5 cm. to the right of the midline. The myocardium was pale and flabby. There was some dilatation of the tricuspid and pulmonic orifices. Examination of the interior leaflet of the tricuspid valve revealed two slightly elevated areas of pin head size, also a grayish-yellow vegetation 2 mm. in diameter, attached to the papillary muscle of this leaflet. When the papillary muscle attached to the aortic cusp of the mitral valve is reflected upward a number of grayish-yellow verrucae are noted. This "pocket lesion" is similar to that described by Libman and Sacks as atypical verrucous endocarditis. Microscopic examination demonstrated round cell infiltration of the epicardium and connective tissue proliferation with collagenous degeneration. In some areas the muscle fibers were large and were separated by an increased amount of connective tissue. Some blood vessels had perivascular round cell infiltration and one thrombosed vessel was noted. In such areas there was a definite increase in fibrous tissue and the muscle fibers were enlarged, elsewhere they were of normal appearance. No Aschoff bodies were found. A section through the endocardium revealed connective tissue proliferation with a mixture of polymorphonuclears and lymphocytes.

The liver weighed 1150 gm. (normal 736 gm.) and extended 3.0 cm. below the ribs in the midclavicular line. The surface was shaggy due to the purulent exudate. The cut tissue was of firm consistency, of brownish-red color, the lobules being prominent. Microscopically the capsule was thickened and there was an infiltrate of polymorphonuclear leucocytes and macrophages. The polygonal cells were compressed due to engorgement of the sinusoids. Marked increase in connective tissue about the trinities with collections of round cells and polymorphonuclears about them was noted, the latter cells infiltrating the parenchyma.

The gall bladder was normal.

The spleen measured 12 x 8 cm. and was 3.4 times its normal weight. The capsule was markedly thickened and the sectioned tissue of a bright red color with a marked increase in connective tissue. Microscopic examination confirmed the fibrosis involving the capsule and showed increased reticulum with areas of fibrosis, the latter areas being similar to the healed lesions of tuberculosis or brucellosis. No giant cells were noted. Vascular changes were noted here as in other organs.

The pancreas was essentially normal.

The adrenals were normal except for round cell collections about blood vessels.

Both kidneys were markedly enlarged, the right and left weighing 160 and 230 grams, two and three times their normal weights. Their capsules stripped with some resistance revealing a smooth gray surface with irregular hemorrhagic areas. The cortex was pale with many beefy hemorrhagic areas. The medullary portions were pale, there being poor differentiation between it and the cortex. Microscopic examination showed many of the glomeruli to be fibrosed and hyalinized with adherence of the tufts to their capsules. The so-called "wire loop" appearance was not observed. A diffuse round cell infiltration involved the parenchyma and seemed localized to a greater degree about the blood vessels whose appearance was similar to that noted in other organs. The tubules exhibited swollen epithelium, their lumen containing albuminous fluid.

No other significant pathological findings were noted. Examination of the brain was not permitted.

In summary, we have evidence of an acute infection of the peritoneum due to the hemolytic streptococcus which may have been secondary to the paracentesis. The changes noted in the spleen are believed to be representative of a healed granulomatous process such as tuberculosis or brucellosis. The renal changes were vascular and those associated with glomerulonephritis. The fibrinoid and collagenous changes in and about the blood vessels plus the myocardial and endocardial involvement are those associated with atypical verrucous endocarditis. The various cardiovascular and renal changes are compatible with those seen in acute disseminated lupus erythematosis. No definite evidence of syphilis was found. It is believed that the positive serology reported was due to lupus erythematosis which not infrequently is responsible for false positive Wassermann and Kahn tests.

PATHOLOGIC DIAGNOSIS

Acute peritonitis
Acute verrucous endocarditis
Acute pericarditis and myocarditis

Generalized vascular and perivascular degenerative changes associated with lupus erythematosis.

Chronic healed granulomatous process involving the spleen

Subacute and chronic glomerulonephritis

Probable Cause of Death: Acute peritonitis and acute disseminated lupus erythematosis.